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Pathogens and Vasculitis

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INTRODUCTION

There are a multitude of studies done regarding the aetiology of vascular accidents. An increasing number of human and animal studies suggest that these vascular sequelae are not only caused by a few infectious agents but could be related to various pathogenic agents, including bacteria, atypical organisms, viruses, fungi and parasites[1].

We believe that the vascular lesions originate in the vasa vasorum of blood vessels and the intimal layers, especially those lesions related to the atypical organisms and various pathogenic agents.

Examples of such accidents are Treponema pallidum (aortitis, retinal vasculitis, cutaneous small-vessel vasculitis), Mycoplasma (IgA vasculitis), cerebral vasculitis and Kawasaki disease. In animal studies, Chlamydia pneumoniae showed inflammatory changes in the aorta and in aortic endothelial cells[1]

The aetiology of vasculitis and it's sequelae are poorly understood. The multitude of vasculitis related disorders in humans vary between unknown causes, bacteria, viruses, fungi, other organisms such as atypical bacteria, Treponema pallidum, Borrelia burgdorferi, etc.

Of the viruses regarding the etiology include HBV,HCV,HIV, VZV, CMV, HTLV-1, EBV, Parvo virus B19, Hantavirus, HSV, Rubella virus and Corona virus[2]. The bacteria implicated include Staphylococcus aureus, Streptococcus, Bartonella henselae, Mycrobacterium tuberculosis, Salmonella, Clostridia, Mycoplasma, etc. Fungi include Candida species, Aspergillus and Coccidioides[2].

It is estimated that about 50% is idiopathic in cutaneous vasculitis syndrome, about 15% due to infections, 20% secondary to inflammatory disease, 15% is drug-induced and about 5% associated with some form of malignancy.[1] Hence, an increasing number of human and animal studies suggests that Vasculitis is caused by a few infectious agents, various pathogenic agents such as viruses, bacteria, fungi, parasites and atypical organisms.

Systemic infection has been proposed to initiate an acute cardiovascular accident (CVA). Those who developed Covid-19 had double the risk of cardiovascular events, while those with severe forms had nearly four times the risk.[2]

Chalmydia pneumoniae and Herpes simplex virus (HSV) cause invasion of the vessel wall and endotheliopathy, acceleration of atherosclerosis through the induction of Cytokines (TNF- alpha, interleukin 2). These are also implicated in chronic inflammation due to multiple infections.[3]

Acute Mycoplasma pneumoniae pneumonia has been reported as a possible cause of diffuse alveolar haemorrhages, adding credence to the fact that haemorrhagic disorders could be secondary to certain microorganisms.[4]

Treponema pallidum is implicated in obliterative inflammation of the vasa vasorum. inflammation and subsequent destruction of the vasa vasorum as the cause of the syphilitic aortitis in tertiary syphilis. The lesion at the vasa vasorum results in ischaemia and weakening of the aortic adventitia, leading to aneurysm formation in the thoracic aorta.

Direct intimal or vasa vasorum infections could be caused by Treponema pallidum, CMV, HSV, Fungi, bacteria and rickettsiae. Direct cell invasion by atypical organisms is thus

[1] *Fioretino DF: Cutaneous vasculitis. J Am Acad Dermatol.2003; 48 (8): 311-40. (Pub Med)*

[2] *Published in Journal Arteriosclerosis, Thrombosis and Vascular Biology. Allayee, H, et al. [2024] Arteriosclerosis, Thrombosis and Vascular Biology. DO1: - 10.1161/ATVBAHA.124.321001.- https://w.w.w.nih.gov*

[3] *Curr Neurol Neurosci Rep.2016 Jan; 16(1): 2.doi: 10.1007/s 11910-015-0602-9*

[4] *J Community Hosp Intern Med Perspect.10; 11 (3); 366-369.doi:10.1080/20009666.2021.1906491*

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implicated.[5] Several mechanisms could be attributed to vasculitis due to infections.[6 7]

Toxin-mediated streptococcal and Staphylococcal disease leading to vascular lesions have been described. [8]

We feel, therefore, that the aetiology of certain "vascular accidents" including aortic aneurysms, intra-cranial haemorrhage, certain CVAs, ischaemic heart disease (IHD) especially in the young, sudden death from certain vascular accidents, pulmonary embolism, spontaneous coronary artery dissection (SCAD), spontaneous coronary artery dissection with leucoencephalopathy (SCADLE) syndrome, etc. may have a common aetiology, at least in a segment of such patients.

Since atypical organisms and certain microbes are implicated in this entity, we suggest that a simple blood test could be used once in two years at least in order to ascertain the high-risk people who could develop these lesions.

At present, rapid sensitive diagnostic methods to detect these organisms are lacking. Besides, viruses and certain bacterial pathogens causing atypical pneumonia such as Mycoplasma pneumoniae, Chlamydia pneumoniae, Chlamydia psittaci, rickettsia, Coxiella burnetti and Legionella species are a few of the pathogens. An atypical organism Chlamydia pneumoniae has been associated with inflammatory conditions in the cardiovascular system and several chronic inflammatory conditions. Further research and trials are warranted to validate such observations. We would like to encourage this research since a simple test could be designed to screen people and identify those at risk for such conditions, especially the young who are susceptible to IHD, CVA,SCAD,SCADLE, pulmonary embolism (PE), transient ischaemic attacks (TIA), etc.

[5] *Dermatol 1999, vol17 (pg 587-90)*
[6] *Immunol,2004, vol.11 (pg 227-31)*
[7] *Clin Exp Rheumatol, 2006, vol 24 (pg 57-81)*
[8] *J Am Acad Dermatol, 1998, vol 39 (pg 383-98)*

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